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Review

Shared and divergent neurocognitive impairments in adult patients with schizophrenia and bipolar disorder: Whither the evidence?

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ABSTRACT

Recent data from genetic and brain imaging studies have urged rethinking of bipolar disorder (BD) and schizophrenia (SCZ) as lying along a continuum of major endogenous psychoses rather than dichotomous disorders. We systematically reviewed extant studies (from January 2000 to July 2015) that directly compared neurocognitive impairments in adults with SCZ and BD. Within 36 included studies, comparable neurocognitive impairments were found in SCZ and BD involving executive functioning, working memory, verbal fluency and motor speed. The extent and severity of neurocognitive impairments in patients with schizoaffective disorder, and BD with psychotic features occupy positions intermediate between SCZ and BD without psychotic features, suggesting spectrum of neurocognitive impairments across psychotic spectrum conditions. Neurocognitive impairments correlated with socio-demographic (lower education), clinical (more hospitalizations, longer duration of illness, negative psychotic symptoms and non-remission status), treatment (antipsychotics, anti-cholinergics) variables and lower psychosocial functioning. The convergent neurocognitive findings in both conditions support a continuum concept of psychotic disorders and further research is needed to clarify common and dissimilar progression of specific neurocognitive impairments longitudinally.

Contents

Abbreviations: ADHD, attention deficit hyperactivity disorder; BACS, the Brief Assessment of Cognition in Schizophrenia; BD, bipolar disorder; BDNF, brain-derived neurotrophic factor; CACNA1C, calcium channel, voltage-dependent, L-type, alpha 1C subunit; COMT, catechol-O-methyl transferase; CPT, continuous performance tasks; CVLT-II, California Verbal Learning Test – Second Edition; D-KEFS, Delis-Kaplan Executive Function System; DLPFC, dorsolateral prefrontal cortex; DSM-5, The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition; DSM-IV, The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; GAF, Global Assessment of Functioning; GWAS, genome wide association studies; HC, healthy controls; ICD-10, International Classification of Diseases and Related Health Problems 10th Revision; IQ, intelligence quotient; NCBI, National Centre of Biotechnology Information; NRGN, neurogranin (protein kinase C substrate, RC3); PFC, prefrontal cortex; PBRM1, polybromo 1; QOL, quality of life; RBANS, Repeatable Battery for the Assessment of Neuropsychological Status; SA, schizoaffective disorder; SANS, Scale for the Assessment of Negative Symptoms; SAPS, Scale for the Assessment of Positive Symptoms; SCZ, schizophrenia; SWCT, Stroop Word-Color Test; TMT A, Trail Making Test A; TMT B, Trail Making Test B; ToH, Tower of Hanoi; WAIS-III, Wechsler Adult Intelligence Scale – Third Edition; WAIS-R, Wechsler Adult Intelligence Scale – Revised Edition; WCST, Wisconsin Card Sorting Test; WHOQOL-BREF, WHO Quality of Life-BREF; WMS, Wechsler Memory Scale; ZNF804A, zinc finger protein 804A.

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1. Introduction

There is increasing evidence to urge some rethinking of major psychiatric disorders such as schizophrenia (SCZ) and bipolar disorder (BD) as lying along a continuum of psychotic disorders (Anderson et al., 2013; Lee et al., 2012). This is in contrast to the Kraepelinian division of major endogenous psychoses into dichotomous disorders of dementia praecox (or SCZ) and manic depressive psychosis (or BD) (Berrios and Beer, 1994; Kraepelin, 1919). First, extant genome wide association studies (GWAS) have identified common susceptibility genes between SCZ and BD such as zinc finger protein 804A (ZNF804A), calcium channel voltage-dependent L-type alpha 1C Subunit (CACNA1C), neurogranin (NRGN) and polybromo 1 (PBRM1) (Bellivier et al., 2013; Craddock and Owen, 2005; Craddock et al., 2006; Goes et al., 2012; Lichenstein et al., 2009; ISC et al., 2009; Williams et al., 2011; Lee et al., 2012). Second, common structural brain abnormalities in gray and white matter brain regions in SCZ and BD have been observed (Anderson et al., 2013; Arnone et al., 2009; De Peri et al., 2012; Ellison-Wright and Bullmore, 2010; Tamminga et al., 2014), particularly in the temporal and frontal regions (Anderson et al., 2013). In addition, heterogeneity in the brain structural and functional neuroimaging findings do not seem to be diagnosis specific (Arnone et al., 2009; Tamminga et al., 2014). Third, clinicians frequently encounter patients with schizoaffective disorder (SA) and BD patients with prominent psychotic symptoms who do not neatly fulfill diagnostic criteria for either category of BD or SCZ (Mancuso et al., 2014). Fourth, dopamine dysregulation has been implicated in both disorders and as such, antipsychotic medications are used for managing symptoms of both SCZ and BD (Cousins et al., 2009; Whitton et al., 2015). A recent study by Tamminga et al. (2014) involving 933 patients with SCZ, SA and psychotic BD probands found that the rates of use of antipsychotic drugs, mood stabilizers and antidepressants were similar across all 3 groups.

Regarding neurocognitive functioning, neurocognitive impairments have been consistently reported to be one of the core symptoms in schizophrenia (SCZ), but a number of recent studies have suggested that patients with bipolar disorder (BD) exhibit significant neurocognitive impairments along the course of their illness (Altshuler et al., 2004; Depp et al., 2007; Ivleva et al., 2012; Kuswanto et al., 2013; Sánchez-Morla et al., 2009; Schretlen et al., 2007). Yet, whilst neurocognitive impairments have been acknowledged to be important clinical findings in SCZ and BD, the extent and severity of shared or discriminant patterns of neurocognitive impairments between SCZ and BD is still being evaluated (Lewandowski et al., 2011b; Murray et al., 2004; Vieta and Phillips, 2007). Previous studies have suggested that patients with SCZ had more severe neurocognitive impairments compared to patients with BD (Lewandowski et al., 2011b; Murray et al., 2004; Vöhringer et al., 2013). However, some studies have also reported that both disorders exhibit comparable degree of neurocognitive impairments in terms of effect sizes across multiple domains (Altshuler et al., 2004; Depp et al., 2007; Ivleva et al., 2012; Kuswanto et al., 2013; Sánchez-Morla et al., 2009; Schretlen et al., 2007). Of note, several studies have observed diminished diagnostic boundary between SCZ and BD when patients with schizoaffective disorder (SA) and psychotic BD were included in the analyses (Amann et al., 2012; Hill et al., 2013; Lewandowski et al., 2011a; McClellan et al., 2004; Reichenberg et al., 2009; Simonsen et al., 2008; Smith et al., 2009) and BD patients with history of psychosis appear to be more cognitively impaired than those without such history in some studies (Martinez-Aran et al., 2008).

The current review thus aims to systematically review extant empirical studies which directly compared the extent and/or severity of neurocognitive impairments between both disorders (including verbal memory, working memory, motor speed, attention, speed of processing and executive functioning). Furthermore, we aim to extend prior work with the inclusion of clinical correlates and finally, evaluation of the level of shared findings in both conditions.

2. Methods

2.1. Literature search

Following guidelines from the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) (Liberati et al., 2009), we searched the National Centre of Biotechnology Information (NCBI), Pubmed/Medline, Scieverse, Scidirect, and Web-of-Science digital databases for empirical studies comparing neurocognitive functioning between patients with SCZ and BD between January 2000 and July 2015. Keywords for the literature search included 'neurocognitive' and 'schizophrenia or schizoaffective disorder' and 'bipolar disorder' (using the algorithm "((neurocognitive) AND (schizophrenia or schizoaffective disorder)) AND bipolar disorder"). We identified potentially useful reports that were then screened as abstracts for meeting inclusion criteria. Promising studies were reviewed as full reports, and their bibliographies were screened for additional references.